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Lack of Acid Key to Thrombi; Enzymes; Angoram Natives Caries-Free

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The following is a transcription of the February 1965 issue of Dr. Royal Lee’s *Applied Trophology* newsletter, originally published by Standard Process Laboratories.

Lack of Acid Held Key to Thrombi

From *Medical World News*, December 4, 1964:

Correction of dietary deficiency may prevent abnormal adhesion of platelets, which Norwegian expert believes is initiating factor in thrombosis.

Most studies of diet and atherosclerosis point up the perils of overnutrition in the prosperous countries of the west.* Taking a different tack, Norway’s Dr. Paul A. Owren stresses a specific deficiency in many diets today, including the widely recommended low-fat diets. The Oslo University physician suggests that a lack of linolenic acid is contributing to the rising incidence of thrombosis and myocardial infarction.

The tendency to thrombosis may be greatly reduced by a daily dosage of linolenic acid, whether in purified form or in linseed oil and soybean oil, according to Dr. Owren and his associates, Drs. Arvid J. Hellem and A.E. Odegaard. Reporting in *Lancet*, they note that linolenic acid, as distinguished from linoleic acid found in some vegetable oils, restores to normal the high adhesiveness of blood platelets in arteriosclerotic patients.

An expert on thrombosis and developer of the Thrombotest (*MWN*, March 17, 1961), Dr. Owren believes that excessive adhesion of blood platelets touches off the chain of chemical reactions leading to a thrombus. He is convinced that the numerous investigators who are trying to link the heightened risk of thrombosis with “hypercoagulability” are pursuing a false trail. “The initial stage in thrombus formation,” he maintains, “is completely independent of plasma coagulation, but depends on platelet function and on plasma factors that are not clotting factors.”

Two Key Variables

Initial platelet aggregation is determined by two key factors, the Oslo investigator says. One is the factor missing in von Willebrand’s disease, which acts together with adenosine diphosphate; the second is factor VIII (antihemophilic factor A). These two variables can “disclose and quantitate” a disposition to thrombus formation, thus serving as warning signals. “A temporary but characteristic rise in the activity of these two factors was found in all the typical clinical conditions that predispose to thrombotic complications, such as the postoperative and postpartum periods, fever reactions, and intravascular hemolysis.”

To counter the heightened tendency to thrombosis, Dr. Owren began looking into diet after he noted that platelet adhesion rose sharply in a patient who had received a blood transfusion from a diabetic donor. At Oslo University’s Institute of Thrombosis Research, founded by Dr. Owren, patients with coronary heart disease were given 100 cc daily of corn oil, cod liver oil, safflower oil, or soybean oil for three days. Soybean oil, rich in linolenic acid, reduced platelet adhesion from about 70 percent to a fairly normal 45 percent. Little or no effect was recorded after administration of the other hypocholesteremic agents.

[Image with caption:] Drs. Owren and Hellem (left) do not agree that plasma coagulation triggers thrombosis.

Similar tests with purified linolenic acid showed a sharp drop in platelet adhesiveness, Dr. Owren notes. “In one patient with coronary artery disease, a single dose of 20 cc of linolenic acid reduced the platelet adhesiveness from 63 percent to about 26 percent in 12 hours. Just 2 cc a day produced a normal level after a week.”

Transfusion Experiment Cited

As evidence that the effect of linolenic acid on platelet adhesiveness is brought about by reduced activity of the anti-Willebrand factor, the Oslo physician cites a transfusion experiment. Platelet-poor plasma from two patients with coronary artery disease sharply increased platelet adhesiveness and cut primary bleeding time in a patient suffering from von Willebrand’s disease.

But after the donors had been pretreated with 30 cc of linseed oil daily for three days, another transfusion of 450 cc plasma had a barely detectable effect. The investigator adds: “The effect of linolenic acid is transitory, and it must be supplied at intervals, preferably daily, to maintain normal platelet adhesiveness.”

Reviewing the changed dietary patterns that may account for a low linolenic acid intake, Dr. Owren calls attention to the decrease in total consumption of carbohydrates in recent decades. He notes, in particular, the dramatic drop in consumption of flours and cereals, which contain a substantial amount of linolenic acid.

While the long-term effect of the acid has yet to be fully documented, “The therapeutic indications seem clear,” Dr. Owren says. “It should be tried in all clinical conditions that are associated with an increased tendency to thrombosis.”

*Perhaps the interpretation would be more appropriate as overeating rather than overnourished. —Editor

Enzymes

We live because we have enzymes. Everything we do is done with enzymes. Without enzymes man could not live. A frequent example that chemistry teachers use to introduce their students to the action of enzymes is the breaking down of the protein in egg whites into its constituent amino acids, i.e., the process of digestion. In the laboratory this requires drastic methods, but in the stomach it proceeds smoothly with a simpler methods—enzymatic action. If I want to break down egg white into its amino acids, I must add 10x its weight of concentrated acids and boil the mixture for 20 hours. Whereas, in the intestinal tract this same breakdown occurs in a couple of hours at body temperature and without the concentrated acid. Furthermore, if I add the stomach lining of a recently killed pig to boiled egg white and keep the mixture at body temperature for a few hours, this breakdown occurs just as it did in the gastrointestinal tract.

Enzymes characteristically have the capacity for enormous selectivity. One ounce of an enzyme preparation from the hog’s stomach will digest 50,000 ounces of boiled egg white in two hours. One ounce of the enzymes, in rennet powder will clot 2,800,000 quarts of milk.

Enzymes are fundamental to life; how we walk, we read, we think, we talk—all we do—is dependent upon enzyme activity.

We get our energy from the sun. Living things cannot use the sun’s energy directly, but only through the mediation of enzymes. Only green plants are able, through their enzymes, to convert the sun’s energy as it falls upon them into chemical energy. The enzymes in the cells of all other living things can then use this stored chemical energy for their daily needs.

All of life is built upon this formula: CO₂ + water + energy (enzymes/of plants)[rightward arrow] sugar + oxygen. The reverse of this formula is battery supplying the energy for the work done by every cell of every living thing: sugar + oxygen (enzymes/in cells) [rightward arrow] CO₂ + water + oxygen. Fortunately, enzymes release the amount of energy needed in a controlled, slow, gradual process.

Each enzyme in the cell has a specific job to do. Every chemical process that takes place in a cell must then have its particular and special enzymes. Since there are literally thousands of these processes, there must be thousands of enzymes in each and every cell.

A milestone in the study of enzymes was that sugar can be fermented into alcohol and carbon dioxide not only by yeast cells but by a watery solution of disintegrated yeast cells in the complete absence of living yeast cells. Hence the name enzyme (literally something *enzyme* in yeast).

Chemists then extracted scores of different enzymes from many different kind of cells, and always the extracts contained protein. So gradually the concept took shape that enzymes were protein. Many German chemists, however, held out for the idea that the protein in these enzyme preparations were impurities and that the enzymes themselves were elusive, smaller molecules present in minute amounts.

James B. Sumner of Cornell University was able to isolate an enzyme in pure form and thus show that it was, in fact, protein. For this work Dr. Sumner was given the Nobel Prize.

Chemists have and are learning about how enzymes act by studying how their action is stopped. A very small amount of cyanide will stop the actions of several enzymes. It is well known that cyanide and iron combine into a closely knit compound that leaves practically no free iron in solution. Cyanide, therefore, stops the action of enzymes in which iron forms an essential constituent by siphoning off the iron. This is why cyanide is such a deadly poison.

(Footnote: The salts of hydrofluoric acid, also poisonous, are known inhibitors of enzymes. —Editor.)

In the April 1965 issue of *Let’s Live*, Melchior T. Dikkers, PhD, Research Biochemist, states: “The most potent enzyme inhibitor known to biochemists is fluorine.”

Then there is another way by which enzymes are inhibited from acting. This is called by the explanatory name —“competitive inhibition.” Here one chemical crowds out another essential one. An illustration from the early research on competitive inhibition was the development of salvarsan and later of sulfanilamide. Para-aminobenzoic acid (PABA) has a formula strikingly like sulfanilamide. The enzymes in bacteria need PABA as an essential nutrient but sulfanilamide is a poison to them. When the vital enzymes in bacteria mistake sulfanilamide for PABA their mechanism stalls, the bacteria then cannot grow and cannot reproduce. Chemists visualize the sulph drugs as competing with PABA for the favors of some enzymes in the bacteria. These are called competitive inhibitors.

Enzymes cannot always do the things that need to be done by themselves. They have to have helpers. Unlike the enzymes themselves these helpers can often take a part in the reaction created by the enzymes.

Some enzymes, for instance, require the presence of very small quantities of a particular metal. Atoms of these metals circulate in the blood and exist in the cells of the body. Metal atoms which help enzymes in their work are called *activators*.

Other enzyme helpers are not metals but are fairly large phosphorus-containing compounds. These are called coenzymes and are related to some of the vitamins.

—*Clinical Physiology*, Vol. 6, No.2, Autumn 1964

Angoram Natives Found Caries-Free

Angoram is a New Guinea village of grass huts situated on the bank of the Sepik River. These tribesmen live off the land entirely, and their diet consists of natural foods, such as meat from crocodiles, possums, snakes, and lizards. Fish, taro root, and yams are the only other source of foods.

A recent study by a dental officer of the Australian New Guinea Health Administration did not find a single cavity in his oral examination of the 110 native villagers. The investigation is to continue principally to determine what substances in the food or soil contributes the most to this ideal situation.



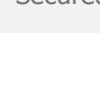
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Selelene River Press is a publishing company specializing in holistic nutrition education. Our philosophy is rooted in the work of Dr. Royal Lee, who showed that vitamin deficiencies, due to processed foods, are responsible for most degenerative disease. From organic farming to food-based vitamins, from raw milk to fermented foods, Dr. Lee set the course for the field of whole food nutrition.

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